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Selective suppression of excessive GluN2C expression rescues early epilepsy in a tuberous sclerosis murine model

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Abstract

Tuberous sclerosis complex (TSC), caused by dominant mutations in either TSC1 or TSC2 tumour suppressor genes is characterized by the presence of brain malformations, the cortical tubers that are thought to contribute to the generation of pharmacoresistant epilepsy. Here we report that tuberless heterozygote Tsc1+/- mice show functional upregulation of cortical GluN2C-containing N-methyl-D-aspartate receptors (NMDARs) in an mTOR-dependent manner and exhibit recurrent, unprovoked seizures during early postnatal life (